scarlet fever seen by them since the sign was discovered.

In the writer's series of 73 cases it was present in every case. The sign has been simultaneous with rash in time of appearance and in about 80% of the cases has lasted from 2 to 3 weeks after the rash has disappeared. In all of the cases seen in our series the sign outlasted the rash by at least 3 to 6 days.

In cases definitely not scarlet fever the sign was seen in 3 cases of hemorrhagic measles, one case of angio-neurotic edema and in one case of Dr. A. A. O'Neill's of dermatitis venenata (poison oak). The measles cases were typical in every way as to history of exposure, mode of onset and appearance on examination. The stripes of the sign were instead of rose red, a deep rusty brown in color and with stippled edges instead of the linear appearance noted in scarlet.

The case of angio-neurotic edema as well as the case of poison oak presented no difficulties in the way of diagnosis and here again the sign was not typical in that there was an edema present, and by reason of structural differences, the skin at the folds merely showed pink lines similar to those which we can ourselves demonstrate by strongly hyperextending our fingers, whereupon the skin folds appear as bright pink lines against the tense white skin of the rest of the palm and palmar surfaces of the digits.

So much for the appearance of the sign in cases definitely not scarlet fever. Now let us complete the survey by considering those cases which may resemble scarlet fever and also the cases of atypical scarlet fever. Three cases of erythematous drug eruption, two due to iodine, and one due to morphine failed to show the sign. Two cases of so-called fish rash also did not present the sign. Two other cases of angio-neurotic edema did not have the sign visible. In fourteen cases of erysipelas the sign was not once seen. Two cases of antitoxin erythema did not show the sign. Two cases of diffuse acute generalized erythema with fever and gastro-intestinal disturbance, but without the usual sore throat or scarlet tongue and no history of contagion were isolated. They did not at any time present the sign and recovered in a few days without any subsequent desquamation.

In a personal communication Dr. Pastia informs the writer of several interesting cases. One is that of a child suffering from hemorrhagic purpura, who, after an injection of peptone, Witte, showed a scarletiniform eruption, including the sign. In this case isolation was justified because not only did the child desquamate but another patient caught scarlet fever from it. Another interesting case is one of erythema multiforme where in the course of a recurring attack the patient was noticed to present the sign. The diagnosis of scarlet was confirmed by the desquamation which followed and was typical of scarlet fever.

In our experience the sign has served with similar success in six cases where the rash was not typical. These cases presented instead of the usual punctiform rash more of the diffuse redness of an erythema scarlatinoides. By virtue of the sign these cases were isolated and proved by their subsequent course that the diagnosis of scarlet fever had been the correct one.

Again quoting from Dr. Pastia's letter he says that "At present in the contagious services in France this sign is given great diagnostic importance in those cases where the eruption is not quite typical and also in retrospective diagnosis, i. e., when the rash has disappeared but the sign remains."

Now to sum up the reasons for accepting this sign as one of diagnostic value: It is an easily identified feature of practically every case of scarlet fever; it is as well marked in the atypical cases as in the typical cases; it persists so that though the rash may not have been observed the diagnosis can yet be made after several days; its occurrence in other diseases has only been noted in such cases as can be easily differentiated from scarlet fever; it has great prophylactic value in those cases where the history or findings or both are otherwise doubtful, especially after the rash has disappeared and desquamation is not visible.

The writer takes this opportunity of expressing his thanks to Dr. A. A. O'Neill for his kindness in placing the material at the Isolation Hospital at his disposal.

Discussion.

Dr. D'Arcy Power, San Francisco: Only two months ago I had a personal experience that is illustrative of the value of this new sign. I was called to a little child seven years of age which suddenly developed a sore throat that looked like tonsilitis. There was a little nausea but nothing specific. As a matter of precaution I looked at the chest and noted what appeared to me a slight scarlatina rash and drew the attention of the parents to it, but was emphatically informed the child's skin always presented this aspect. The picture by itself was not sufficient to justify a diagnosis of scarlatina and I should undoubtedly not have done so had I not remembered reading of the Pastia sign and on looking at the skin folds in the arm, noted it fully present. I stuck to my diagnosis and was justified by the later history of the case.

CLINICAL ASPECTS OF UROSEPSIS.*

By M. KROTOSZYNER, M. D., San Francisco.

Various are the names and manifold the classifications under which infections of the urinary tract and systemic toxemias, due to one or more septic foci in the uropoetic system, are recorded in the literature. Guiteras' exhaustive text-book, for instance, which represents the most modern views on matters urological, deals with the subject under the different headings of "Urinary fever, Catheter fever, Urinary infection." Watson and Cunningham, to quote one more authoritative standard urological work, devote a brief chapter to "urethral shock or chill—urinary infection or urinary fever."

* Read before the Forty-Second Annual Meeting of the State Society, Del Monte, April, 1912.

Similarly confusing is the classification under which this subject is treated in the rather scant contributions of our periodical literature.

It appeared, therefore, a grateful task to hit upon a name, which would embrace the subject in all its manifestations and phases, and, at the same time, be brief and suggestive of its most important features, viz: urinary infection and intoxication. After careful deliberation and an exhaustive search of the literature, pertaining to this subject, the name "Urosepsis" appeared to be the most appropriate, the simplest and the briefest technical term.

This name is, at present, given to a systemic septic condition, originating in the urinary tract and presenting the picture of a chronic urinary intoxication. The systemic toxemias caused by an ascending infection, due to stagnation of urine in the bladder, ureters and renal pelvis, or by insufficient renal elimination and absorption of toxins, result, as a rule, in the clinically well-defined septicemic condition, which is characterized by digestive disturbances, loss of appetite, a heavily coated tongue, marked general debility and irregular temperatures (septic fever). This characteristic complex of symptoms which is designated by good authority as "Urosepsis" is very frequently observed in old prostatics with ascending infectious processes of the upper urinary tract (ascending pyelitis, pyelonephritis and pyonephrosis). At postmortem examination these lesions, in most instances, represent the fatal cause or demonstrate the cause of failure of a belated prostatectomy. An equally grave form of urinary toxemia, on the other hand, may occur without renal involvement and as a result of absorption of urinary or bacterial toxins from one or more septic foci in the prostate or bladder. I have seen several cases of typical and prolonged urosepsis in elderly prostatics, where, on the autopsy-table, no or such insignificant renal lesions were found, that they alone could not be held responsible for the fatal outcome. It is, furthermore, obvious that a grave and frequently fatal toxemia may occur on the basis of a trauma of the urinary tract, through which urine may escape into the surrounding tissue, where it gradually, through stagnation and decomposition, will exert its toxic properties. Clinically these pathological conditions offer like or similar aspects and etiologically they belong to the same class on account of their origin from one or more infecting foci of the urinary tract.

We are, therefore, justified in combining all these infections and toxic conditions of the urinary tract under the collective term of "urosepsis" differentiating them with regard to their special or topical etiological features as renal, vesical, traumatic and so forth.

The most important cause of traumatic urosepsis is, obviously, instrumentation, and modern urologists are aware of the important role which it plays in producing local and systemic urinary infections. While it is conceded that by adherence to strict asepsis such infections can be numerically reduced to a minimum, we, nevertheless, occasionally encounter cases, in which the simple act of urethral catheterization, performed under the most rigid aseptic precautions, is followed by a grave and even fatal systemic toxemia.

A diabetic of 62 entered the hospital with complete retention due to acute prostatic congestion. Apex of bladder at navel. Gradual evacuation of bladder by means of a small-calibred soft-rubber catheter under laparatomy-asepsis. The same or similar aseptic measures are observed at each consecutive catherization. Symptoms of typical renal urosepsis set in within 48 hours from the first catheterization and the patient succumbs, within a week, to his ascending infection.

It is, on the other hand, a well known fact, that a notoriously "septic" catheterism must not necessarily be followed by untoward symptoms and even in these later days of widely spread knowledge of the value of asepsis, there are still many elderly prostatics immunedly using their catheters with very insufficient or no means of sterilization. Such and similar observations, which occur in the daily practice of every experienced urologist, prove that local and systemic urinary infections are not alone due to a flaw in the technic or asepsis of instrumentation. The real cause of infection lies, in all probability, in the existence of a *locus minoris resistentiæ* somewhere in the lower urinary tract, like a slight abrasion or a minute trauma of the mucosa, through which the entrance and propagation of infecting microorganisms takes place. The toxic material is not, necessarily, brought into the bladder by means of instruments, the introduction of which merely hastens the outbreak and generalization of the preexisting septic focus. In this way the not very rare instances of acute and, occasionally, prolonged febrile reactions to delicately and most carefully performed instrumentation (traumatic or instrumental urosepsis) find their plausible explanation.

A married man of 42 had acquired an acute gonorrhea and was seen a day after an attempt to inject an astringent fluid dispensed by a druggist. Patient is slightly delirious, shows great prostration, heavily coated tongue, high fever. This condition gradually abated within two weeks. The patient came, many months later, under treatment for a chronic urethritis complicated by a stricture. Each attempt of instrumentation (steel-sound) is followed by high fever and malaise lasting several days. These attacks can be reduced in intensity but are not entirely checked by prophylactic large doses of quinine.

An urinary infection, in reality, does not as often, as it was thought heretofore, take place through mechanical interference or by contact-contamination, or ascending proliferation. Through the investigations of Guyon, Posner and Lewin, Wildholz, Brewer and others we know to-day that an urinary infection, in most instances, occurs by the hematogenous route. The kidneys are the dumping-place for the waste-material of metabolism, where these undesirable end-products remain longer than at other parts of the organism. If, then, by way of the circulation, pathogenic bacteria are thrown into the system, they will find in either of the kidneys with an insignificant lesion, a fertile soil for habitation and spread. The classical instance for this mode of infection is furnished by renal tuberculosis, which invariably represents an hemotogeneous and not, as is still tenaciously held in many quarters, an ascending infection on the basis of vesical tuberculosis.

A man of 32 was referred to me some years ago by a prominent genito-urinary specialist of New York, who had advised his client to remove to California for climatic purposes. Very frequent and distressing micturition, many tubercle bacilli in the urine-sediment. Cystoscopy reveals many ulcerations and tubercles. On account of the patient's extreme suffering and, after due counsel, the bladder-wall was scraped and the viscus drained through a suprapubic cystotomy. The patient rapidly becomes septic, delirious and uremic. Exitus within a few weeks from operation.

At the time of this observation Israel and Nitze still sounded their authoritative warnings against ureteral catheterization in urinary tuberculosis on account of the theoretically adduced danger of conveying tubercular virus from the infected bladder to a healthy kidney. To-day we know that the current of infection runs downward and practical experience has demonstrated in innumerable instances the innocuousness of bilateral ureteral catheterization in renal tuberculosis through a bladder that presents many ulcerated tubercular foci.

Other examples of hematogenous infection are the occurrence of a calculous pyelitis in the course of an aseptic renal calculosis or the metastatic renal infections from remote suppurative or inflammatory foci (panaritium, tonsillitis, etc.). In this connection the much-discussed coli infections of the urinary tract, probably caused by migration of normal intestinal inhabitants into the bladder or kidney deserve mention. It is proved that the so-called catheter-fever, which formerly was thought to be caused by shock, irritation of certain nerve centers, malaria, etc., is in most instances due to coli-infection. For the more protracted forms of traumatic urosepsis with a remittent fever and frequent attacks of chills, followed by profuse sweating we will justly assume a streptococcus-invasion. Such cases may take a favorable turn, but may occasionally terminate fatally.

A girl of 18 entered the hospital for incision of a suppurative gonorrheal bartholinitis. Complete euphoria after operation, therefore the patient leaves the hospital in spite of her profuse vaginal and urethral discharge. Reenters 2 weeks later with acute pelviperitonitis, cystitis and pyelitis. Urine-sediment shows B. Coli and streptococci in pure-culture. Exitus occurs within ten days under symptoms of cardiac insufficiency with repeated attacks of collapse, vomiting, singultus, dry tongue, delirium.

The gravest form of urosepsis, though, is the one which prevents either no or only moderate elevations of temperature and which is characterized, from the beginning, by digestive disturbances (abhorrence of food, especially of meat) distressing thirst, disturbances of deglutition and low or insufficient urine-excretion. This form represents a combination of urinary infection and systemic intoxication through the normal or decomposed urine. The toxicity of normal urine is, since the investigations of Bouehard and his pupils, a well-established fact. If the urine stagnates in the bladder and, through backward pressure upon the ureters and renal pelvis, interferes with the excretory function of the kidneys, absorption of urine-toxins and chronic urine-intoxication ensues. The early recognition of this condition, and the timely establishment of a free urinary flow (elimination of urine-toxins) by mechanical means (suprapubic fistula, prostatectomy, etc.), are most grateful therapeutical procedures.

A man of 64, in the third stage of prostatism (dribbling from overdistended bladder) shows marked symptoms of urosepsis. Irregular low fever-curve, dry tongue, continuous thirst, lack of appetite, slight mental aberrations, and marked cachexia. Drainage through a suprapublic fistula, under local anesthesia, results in improvement of uroseptic symptoms; therefore, 4 weeks later, enucleation of protruding median prostatic lobe under spinal anesthesia. Pathological report: Carcinoma of prostate. After a brief period of improvement the patient gradually relapses into a semi-comatous condition with marked uroseptic and uremic symptoms. Exitus.

A prostatic of 74, with advanced urosepsis and almost complete retention, continues in this condition, after suprapublic prostatectomy under spinal anesthesia, for many weeks. Various stages of exacerbation and remission of uroseptic symptoms (irregular temperature, digestional disturbance, slight coma). Gradually uroseptic symptoms abate. complete recovery.

In conclusion I beg to present the following classification of infections and toxic conditions of the urinary tract, which appears to be in accord with our modern views on that subject:

It is proposed to combine all these conditions under the collective term: "urosepsis."

According to the clinical course urosepsis may be divided in the acute, subacute and chronic form.

According to the topical etiology a urethral (prostatic) vesical and renal form may be differentiated.

Other prominent types of urosepsis are the traumatic (instrumental) and bacterial (coli, streptococcus, etc.), forms.